A note on a paper by Erik Volz: SIR dynamics in random networks

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Abstract

Recent work by Erik Volz [12] has shown how to calculate the growth and eventual decay of an SIR epidemic on a static random network, assuming infection and recovery each happen at constant rates. This calculation allows us to account for effects due to heterogeneity in degree that are neglected in the standard mass-action SIR equations. In this note we offer an alternate derivation which arrives at a simpler — though equivalent — system of governing equations to that of Volz. This new derivation is more closely connected to the underlying physical processes, and the resulting equations are of comparable complexity to the mass-action SIR equations. We further show that earlier derivations of the final size of epidemics on networks can be reproduced using the same approach, thereby providing a common framework for calculating both the dynamics and the final size of an epidemic spreading on a random network.

1 Introduction

Infectious diseases are constrained to spread along the contacts of a population. Mathematical models investigating epidemics typically assume that the contacts occur through mass action mixing [6, 1]. However true populations violate some mass-action assumptions in a manner affecting the epidemic dynamics. Recently a number of investigations have been performed using random networks [9, 5, 8, 11, 7] which allow for a better accounting of mixing in the population.

Unlike mass-action models, random networks allow for the number of contacts individuals have to remain bounded as the population size increases. Thus once an individual infects a contact, the number of available contacts to infect decreases by a non-negligible amount. Random networks also allow for more accurate representation of heterogeneities in the number of contacts compared with mass-action models. In a population with heterogeneous contact levels,

individuals with more contacts are preferentially infected early in the epidemic (and in turn cause more infections), while at the end of the epidemic the remaining susceptibles tend to have fewer contacts.

A number of analytic results have been found for epidemic probability or size in random networks, but with only a few exceptions (notably [11, 12]), no analytic attention has been paid to the dynamics of the growth in networks. However, some attempts have been made using *pair approximations* which track the number of joined pairs of individuals with k_1 contacts and k_2 contacts in each infection state [3] (assuming infection and recovery occur at constant rates). For a network with n different degrees, such a model results in $\mathcal{O}(n^2)$ coupled differential equations.

Recent work by [12] has shown that it is possible to investigate the dynamics of epidemic spread on Configuration Model networks (described below) using a coupled system of only three ODEs (again assuming infection and recovery occur at constant rates). The resulting system has many nonlinear terms, but the number of equations does not grow with the number of different degrees. In this note we derive a single differential equation that can capture the dynamics with only a single higher order term. The framework we develop to calculate the dynamics can also be applied to predicting the final size of an epidemic in a concise way. We reproduce earlier results in this context.

Although our results are equivalent to pre-existing results, we place previous calculations of epidemic size and epidemic dynamics into a common framework. The equations we derive are simpler, and the terms in the equations are more easily interpreted. The resulting calculations for the numbers of susceptible, infected, and recovered individuals are of comparable complexity to the standard mass-action SIR equations, but allow for more realistic population interactions.

In section 2 we develop the framework for the later sections. In section 3 we apply this framework to calculating the time course of an epidemic. In section 4 we apply this framework to calculating the final size of an epidemic. Finally in section 5 we discuss the significance of these calculations.

2 The framework

We represent the population by a network. Each individual is thought of as a node joined to other nodes by edges through which disease can spread. We use Configuration Model (CM) networks [10] to model the population. To generate a CM network, the *degree* or number of edges of each node, k, is assigned with probability $P(k)$ based on a given degree distribution. If the sum of degrees is odd, all degrees are reassigned until the sum is even. Then each node is placed into a list with repetition equal to its degree, the list is randomized, and each node in position $2n$ ($n = 0, 1, \ldots$) is connected with the node in position $2n + 1$. The resulting network constitutes a uniform choice from the networks with the given degree distribution. In general the network may have self-loops or repeated edges. For degree distributions with finite mean, the impact of this

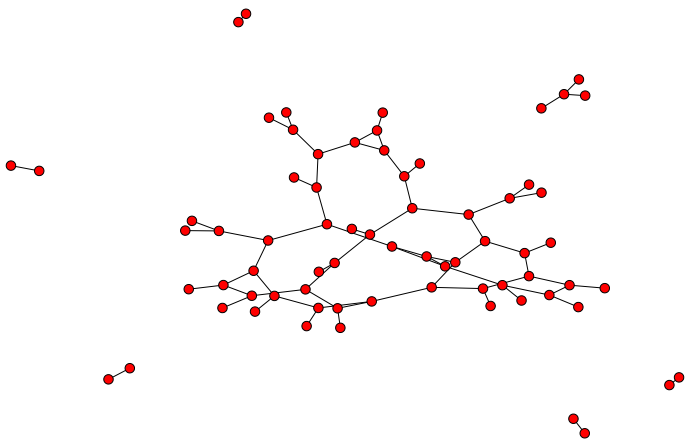

Figure 1: A sample Configuration Model network with 70 nodes. The degree distribution is chosen such that $P(3) = 0.5$ and $P(1) = 0.5$. Thus $\psi(x) = (x^3 + x)/2$.

effect is negligible in sufficiently large networks and we ignore it. We define

$$\psi(x) = \sum_{k=0}^{\infty} P(k)x^k \,,$$

the probability generating function of the degree distribution. Note that $\psi'(1) = \langle K \rangle$ is the average degree. An example CM network is shown in figure 1. For many important distributions, ψ takes a simple form; for example, a Poisson distribution with parameter λ has $\psi(x) = e^{\lambda(x-1)}$.

Nodes in the network are assigned to one of three classes: susceptible, infected, or recovered. We denote the fraction of the population in each class by S, I, and R respectively. A susceptible node becomes infected at rate $n\beta$ where n is the number of infected neighbors it has. Once infected, a node recovers at rate γ. A recovered node plays no further role in the spread. Typically an outbreak is initiated with a single randomly chosen infected indvidual in an otherwise susceptible population.

We define an *infectious contact* from v to its neighbor u to be a contact when v is infectious that would cause infection of u if u were susceptible. Physically this is the transmission of an infectious dose from v to u. An individual can cause infectious contact only when infected. However, an individual can receive an infectious contact regardless of his/her state, and so an infectious contact does not necessarily lead to infection.

We use θ as a measure of the probability that a random edge has not transmitted an infectious contact. Its precise definition is subtle, but important. To define θ, we choose an edge uniformly from all edges. We then choose a direction

for that edge, say from v to u. We refer to v as the *base* and u as the *target*. We modify the spread of the disease by disallowing infectious contacts from u to v. Then $\theta(\infty)$ is the probability that there is never an infectious contact from v to u, while $\theta(t)$ is the probability that at time t there has not been infectious contact from v to u. If we did not disallow infection from the target then an infection of u from some other source would in turn make infection of v more likely, which in turn makes infectious contact from v to u more likely, and so transmission along different edges to the same target would not be independent, thereby complicating the analysis.

Under the assumption that the spread is deterministic, the cumulative size of an epidemic at a given time is equal to the probability a randomly chosen node has been infected. Disallowing infection from that single randomly chosen node may impact the dynamics *after* that node is infected, but it does not modify the probability that that single node has become infected. Consequently, to calculate the size at a given time, it suffices to calculate the probability a randomly chosen node that cannot infect its neighbors has been infected, or alternately, is still susceptible.

3 Dynamics

To calculate the dynamics, we calculate the fraction of the population that has not yet been infected. To do this, we look at the probability that a randomly chosen node is not yet infected at time t. We choose a random target u and disallow infection from u to all of its neighbors. Using θ as defined above, if the degree of u is k, then the probability that u is still susceptible is $\theta(t)^k$. Thus the fraction of susceptibles is

$$S(t) = \sum_{k=0}^{\infty} P(k)\theta(t)^k = \psi(\theta(t))\,. \tag{1}$$

To calculate the rate of change of θ, we will need to know how many of those edges that have not transmitted an infectious contact have the opportunity to transmit infection at any given time. That is, we need to know what proportion of all edges have not had an infectious contact but come from an infected base node. We set ϕ to be the probability that the base node of an edge is infected but the edge has not transmitted infection (assuming as for θ that the target node does not cause infection). Those edges which satisfy the definition for ϕ are a subset of those which satisfy the definition for θ.

We derive coupled differential equations for θ and ϕ. The rate of change in the probability a random edge has not transmitted infection is equal to the rate at which infection crosses edges

$$\dot{\theta} = -\beta\phi\,. \tag{2}$$

An edge no longer satisfies the definition for ϕ when infection crosses the edge or when the base node recovers. An edge from v to u begins to satisfy the definition

if v becomes infectious. The rate at which neighbors become infectious matches the rate at which neighbors stop being susceptible. We use $h(t)$ to denote the probability that a neighbor is susceptible, so $\dot{\phi} = -(\beta+\gamma)\phi - (d/dt)h(t)$.

We now find $h(t)$. A node is more likely to be a neighbor if it has more contacts [4], and so the probability the neighbor has degree k is $kP(k)/\langle K\rangle$. The neighbor can only be infected by an edge other than the one from the target node. Thus

$$h(t) = \frac{\sum_{k=0}^{\infty} kP(k)\theta^{k-1}}{\langle K\rangle} = \frac{\psi'(\theta)}{\psi'(1)}\,.$$

Thus the neighbor becomes infectious at rate $-(d/dt)h(t) = \beta\phi\psi''(\theta)/\psi'(1)$. We finally get

$$\dot{\phi} = \left[-\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)}\right]\phi\,. \tag{3}$$

In fact, we can integrate this equation using (2) to get

$$\phi = 1 - (1-\theta) - \frac{\gamma}{\beta}(1-\theta) - \frac{\psi'(\theta)}{\psi'(1)}\,.$$

The term $1-\theta$ represents the probability the edge has transmitted an infectious contact, the term $(\gamma/\beta)(1-\theta)$ represents the probability that the base node has been infected but recovered without an infectious contact, and $\psi'(\theta)/\psi'(1)$ represents the probability that the base node is still susceptible. The complement of all such edges is exactly those edges which have not transmitted infection but connect to an infected base node. Consequently we arrive at

$$\dot{\theta} = -\beta\theta + \gamma(1-\theta) + \beta\frac{\psi'(\theta)}{\psi'(1)}\,. \tag{4}$$

The epidemiological quantity of interest is only rarely the proportion of edges which have or have not transmitted infection, but rather it is usually the values of S, I, and R. We can calculate $S(t) = \psi(\theta(t))$ directly. It is not difficult to show that $\dot{R} = \gamma I$, and conservation of individuals gives $I = 1 - S - R$. Consequently, we can augment (4) with

$$\begin{aligned}\dot{R} &= \gamma I\,,\\ S &= \psi(\theta)\,,\\ I &= 1 - R - S\,.\end{aligned}$$

to find S, I, and R.

In order to solve our equations, we need to find appropriate initial conditions. At the earliest stages, the outbreak grows stochastically, and so the deterministic equations are not yet appropriate. If an epidemic occurs, eventually the outbreak infects a large number of nodes and then behaves effectively deterministically. In a sufficiently large population we can assume that deterministic behavior begins while the proportion infected is still small compared to the population.

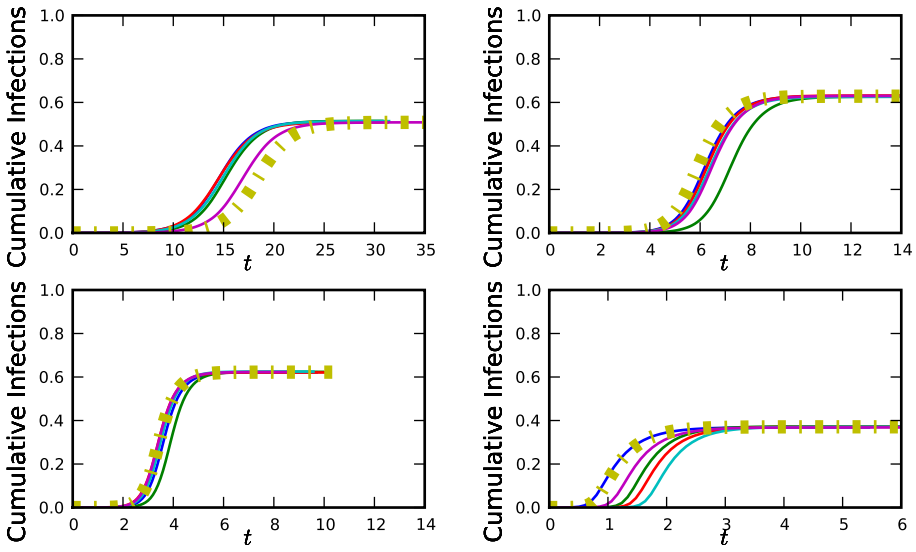

Figure 2: Plots of cumulative infections $I + R$ against time. Predicted epidemic dynamics (thick, broken curves) and final sizes compared with simulations (solid curves) in CM networks of 500 000 individuals with $\beta = 1.3$ and $\gamma = 2$. (a) Every node has degree 4. (b) Poisson degree distribution of mean 4. (c) A bimodal distribution: $P(1) = 5/12$, $P(2) = 1/12$, $P(6) = 1/12$, and $P(7) = 5/12$. (d) A truncated powerlaw with $P(k) \propto e^{-k/40}k^{-2.5}$.

Once the stochastic phase is over, we have $\theta = 1 - \epsilon$ with $\epsilon \ll 1$. At early time $\epsilon \propto \exp[(-\beta - \gamma + \beta\psi''(1)/\psi'(1))t]$ unless $\psi''(1)$ is infinite (which corresponds to an infinite variance in the degree distribution such as occurs in some power-law distributions). For simplicity we assume the $\psi''(1)$ is finite (if it were not, growth would not be exponential initially and this calculation would require more attention). We define $t = 0$ to correspond to a time when the epidemic is sufficiently large that the outbreak proceeds deterministically, but the proportion affected is still small. From the value of θ we can easily calculate $S(t) = \psi(\theta(t))$, and thus we can also calculate $I + R$.

To distinguish the number of current infections (I) from recovered infections (R) requires somewhat more effort. To find the early behavior for R, we note that I and ϵ are linearly related at early time, so that $I \propto \exp[(-\beta - \gamma + \beta\psi''(1)/\psi'(1))t]$. Then $\dot{R} = \gamma I$ gives $R = \gamma I/[-\beta - \gamma + \beta\psi''(1)/\psi'(1)]$. Combined with $I + R = 1 - S = 1 - \psi(1 - \epsilon)$ this gives R at $t = 0$.

We show a comparison of simulation with results calculated using equation (4) in figure 2. The results show good agreement, except for time shifts resulting from stochastic effects in the simulations while the outbreak size is still small.

3.1 Discussion

Equation (4) contrasts with the original system of [12] which uses three equations. In addition to the variable θ, the system of [12] uses $p_I = \phi/\theta$ (the probability that an edge is connected to an infected node given that it has not transmitted infection to the target node) in place of ϕ and an additional variable p_S (the probability that an edge is connected to a susceptible node given that it has not transmitted infection to the target node):

$$\begin{aligned}\dot{\theta} &= -\beta p_I \theta\,,\\ \dot{p}_I &= p_I\left[\beta p_S \theta \frac{\psi''(\theta)}{\psi'(\theta)} - (\beta+\gamma) + \beta p_I\right]\,,\\ \dot{p}_S &= \beta p_S p_I\left[1 - \theta\frac{\psi''(\theta)}{\psi'(\theta)}\right]\,.\end{aligned}$$

We have replaced this system by the single equation (4) with only one higher order term. To see that these systems are equivalent, we note the $\dot{p}_S$ equation can be eliminated by observing that the probability the neighbor has not been infected is $\psi'(\theta)/\psi'(1)$ and so $p_S = \psi'(\theta)/\theta\psi'(1)$. Equation (3) can be modified by using $\psi'(1) = \psi'(\theta)/\theta p_S$ and $\phi = \theta p_I$ to arrive at the same $\dot{p}_I$ equation.

4 Final epidemic size

We now reproduce some of the earliest results for epidemics on networks [8, 9, 2] by calculating the final size of epidemics (under the assumption that the outbreak does not die out during the stochastic phase). We can find this by solving equation (4) for $\dot{\theta} = 0$. However, this approach is unnecesarilly specific and we can easily generalize to disease processes that do not depend on constant infection and recovery rates by calculating $\theta(\infty)$ directly rather than through equations for the intermediate dynamics. To simplify notation in this section we use θ_∞ to represent $\theta(\infty)$ as we are not interested in the epidemic state at intermediate time.

To calculate the epidemic size, we look for the probability that a randomly chosen node u is never infected. If a node has degree k, then the probability that it is never infected is θ_∞^k. From this we get

$$S(\infty) = \sum_{k=0}^{\infty} P(k)\theta_\infty^k = \psi(\theta_\infty)\,. \tag{5}$$

We must calculate θ_∞. We set $T = \int_0^\infty \gamma e^{-\gamma\tau}(1 - e^{-\beta\tau})\,d\tau = \beta/(\gamma+\beta)$. This is the probability that a randomly chosen neighbor has an infectious contact with u given that the neighbor becomes infected. If h is the probability that the neighbor does not become infected (given that u does not transmit infection), the probability of infectious contact is $T(1-h)$. Thus the probability of not transmiting is

$$\theta_\infty = 1 - T(1-h) = 1 - T + Th\,.$$

An argument in the previous section shows that $h = \psi'(\theta_\infty)/\psi'(1)$, and so θ_∞ solves the implicit relation

$$\theta_\infty = 1 - T + T\frac{\psi'(\theta_\infty)}{\psi'(1)}\,. \tag{6}$$

Using (5) and (6) together gives $S(\infty)$, and the final size of an epidemic is simply $1 - S(\infty)$.

Note that the ability of a base node to infect a neighbor depends on duration of infection and whether the base node becomes infected. Consequently, infectious contacts along different edges out of the same node are not independent events (they both depend on the base node's properties). However, this does not affect our calculations because infectious contacts along different edges into the same node are independent events. If there were variation in susceptibility, more work would be needed [8]. Also the independence assumption will fail if short cycles are not negligible because infection of one neighbor is correlated with infection of another.

5 Discussion

We have shown that calculations for both the final size and the dynamics of an epidemic on a random network can be placed into a common framework. This framework allows us to simplify previous calculations of the dynamics [12]. Our calculations match closely to simulations, except for time shifts that result from stochastic effects when the infected population is still small. Our model is of similar complexity to the standard mass-action SIR equations.

The assumption that the network is a Configuration Model network is central to this derivation. If there is a tendancy for high degree individuals to preferentially contact high degree individuals, these approaches do not directly apply. Similarly the presence of many short cycles will also affect these calculations. When a short cycle exists, whether or not one neighbor of the target node is still susceptible may no longer be independent of whether another neighbor is still susceptible.

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